



STIC Search Report

Biotech-Chem Library

STIC Database Tracking Number: 175328

TO: Kevin Weddington
Location: REM-3A65/3C70
Art Unit: 1614
Thursday, January 05, 2006

Case Serial Number: 10/707087

From: Mary Jane Ruhl
Location: Biotech-Chem Library
Remsen 1-A-62
Phone: 571-272-2524

maryjane.ruhl@uspto.gov

Search Notes

Examiner Weddington,

Here are the results for your recent search request.

Please feel free to contact me if you have any questions about these results.

Thank you for using STIC services. We appreciate the opportunity to serve you.

Sincerely,

Mary Jane Ruhl
Technical Information Specialist
STIC
Remsen 1-A-62
Ext. 22524



STIC SEARCH RESULTS FEEDBACK FORM

Biotech-Chem Library

Questions about the scope or the results of the search? Contact ***the searcher or contact:***

Mary Hale, Information Branch Supervisor
Remsen Bldg. 01 D86
571-272-2507

Voluntary Results Feedback Form

➤ I am an examiner in Workgroup: Example: 1610

➤ Relevant prior art **found**, search results used as follows:

- ☐ 102 rejection
- ☐ 103 rejection
- ☐ Cited as being of interest.
- ☐ Helped examiner better understand the invention.
- ☐ Helped examiner better understand the state of the art in their technology.

Types of relevant prior art found:

- ☐ Foreign Patent(s)
- ☐ Non-Patent Literature
(journal articles, conference proceedings, new product announcements etc.)

➤ Relevant prior art **not found**:

- ☐ Results verified the lack of relevant prior art (helped determine patentability).
- ☐ Results were not useful in determining patentability or understanding the invention.

Comments:

Drop off or send completed forms to STIC-Biotech-Chem Library Remsen Bldg.



REM- 3070

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175328

ACCESS DB #

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Scientific and Technical Information Center

SEARCH REQUEST FORM

Requester's Full Name: K. Weddington Examiner #: 68082 Date: 12-29-05
Art Unit: 1614 Phone Number: 2-0587 Serial Number: 10/707,087
Location (Bldg/Room#): 3A65 (Mailbox #): _____ Results Format Preferred (circle): PAPER DISK

To ensure an efficient and quality search, please attach a copy of the cover sheet, claims, and abstract or fill out the following:

Title of Invention: _____

Inventors (please provide full names): Muhammed Majeed; Rajinder Kumar Bammi;
Natarajan Sankaran; Rajendern Ramanujan; Satyan Kalkunte Seshadri

Earliest Priority Date: _____

Search Topic:

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc., if known.

For Sequence Searches Only Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.

Theating obesity or weight loss with
forskolin
iso forskolin
deacetyl forskolin

* Also search the plant *Coleus forskohii* contains
Forskolin

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Searcher: _____

Searcher Phone #: _____

Searcher Location: _____

Date Searcher Picked Up: _____

Date Completed: _____

Searcher Prep & Review Time: _____

Online Time: _____

Type of Search

____ NA Sequence (#)

____ AA Sequence (#)

____ Structure (#)

____ Bibliographic

____ Litigation

____ Fulltext

____ Other

Vendors and cost where applicable

____ STN _____ Dialog

____ Questel/Orbit _____ Lexis/Nexis

____ Westlaw _____ WWW/Internet

____ In-house sequence systems

____ Commercial _____ Oligomer _____ Score/Length
____ Interference _____ SPDI _____ Encode/Transl
____ Other (specify)

=> d his ful

(FILE 'HOME' ENTERED AT 13:28:26 ON 05 JAN 2006)

FILE 'HCAPLUS' ENTERED AT 13:30:06 ON 05 JAN 2006

E MAJEED MUHAMMED/AU
L1 41 SEA ABB=ON ("MAJEED MOHAMMED A"/AU OR "MAJEED MUHAMMAD
IRFAN"/AU OR "MAJEED MUHAMMED"/AU OR "MAJEED MUHAMMED A"/AU)
E BAMMI RAJINDER KUMAR/AU
L2 2 SEA ABB=ON "BAMMI R K"/AU
E SANKARAN NATARAJAN/AU
E SANKARAN N/AU
L3 27 SEA ABB=ON ("SANKARAN N"/AU OR "SANKARAN N B"/AU OR "SANKARAN
NARAYANAN"/AU)
E RAMANUJAM RAJENDRAN/AU
L4 13 SEA ABB=ON RAMANUJAM R/AU
L5 4 SEA ABB=ON "RAMANUJAM R A"/AU
L6 17 SEA ABB=ON L4 OR L5
E SESHADRI S/AU
L7 264 SEA ABB=ON ("SESHADRI S"/AU OR "SESHADRI S G"/AU OR "SESHADRI
S J"/AU OR "SESHADRI S K"/AU OR "SESHADRI S KRISHNAMOORTHY"/AU)
E PRAKASH SUBBALAKSHMI/AU
L8 8 SEA ABB=ON "PRAKASH SUBBALAKSHMI"/AU
L9 0 SEA ABB=ON L1 AND L2 AND L3 AND L4 AND L6 AND L7 AND L8
L10 352 SEA ABB=ON L1 OR L2 OR L3 OR L4 OR L6 OR L7 OR L8
L11 0 SEA ABB=ON L10 AND ?LABDANE?
L12 4 SEA ABB=ON L10 AND ?TERPENE?
L13 ANALYZE L12 1-4 CT : 61 TERMS

FILE 'REGISTRY' ENTERED AT 13:35:21 ON 05 JAN 2006

L14 3 SEA ABB=ON (FORSKOLIN OR ISOFORSKOLIN OR DEACETYLFORSKOLIN)/CN
E COLEUS FORSKOHII/CN

FILE 'HCAPLUS' ENTERED AT 13:35:56 ON 05 JAN 2006

L15 16023 SEA ABB=ON (L14 OR FORSKOLIN OR ISOFORSKOLIN OR DEACETYLFORSKO
LIN OR ?COLEUS?(W)?FORSKOHII)
L16 75764 SEA ABB=ON OBESITY+ALL OR WEIGHT LOSS+ALL
L17 89 SEA ABB=ON L15 AND L16
L18 72 SEA ABB=ON L17 AND (PRD<20031120 OR PD<20031120)
L19 1 SEA ABB=ON L18 AND (?METHOD?(L)?PREP?)
L20 32 SEA ABB=ON L18 AND (?PRODUC? OR ?PREP? OR ?SYNTH?)
L21 4 SEA ABB=ON L20 AND ?METHOD? *4 cit's from CAPLUS*

FILE 'MEDLINE, BIOSIS, EMBASE, JAPIO, JICST-EPLUS' ENTERED AT 13:38:34 ON
05 JAN 2006

L22 10 SEA ABB=ON L21
L23 9 DUP REMOV L22 (1 DUPLICATE REMOVED) *9 cit's from database*

FILE 'USPATFULL' ENTERED AT 13:40:08 ON 05 JAN 2006

L24 889 SEA ABB=ON L20 AND ?METHOD?
L25 876 SEA ABB=ON L24 AND ?METHOD?(L)?PREP?
L26 19 SEA ABB=ON L25 AND ?LEAN?(W)?BODY?(W)?MASS? *19 cit's from
USPatFull*

FILE HOME

FILE HCAPLUS

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FILE COVERS 1907 - 5 Jan 2006 VOL 144 ISS 2
FILE LAST UPDATED: 4 Jan 2006 (20060104/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE REGISTRY

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 4 JAN 2006 HIGHEST RN 871209-00-6
DICTIONARY FILE UPDATES: 4 JAN 2006 HIGHEST RN 871209-00-6

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH JULY 14, 2005

Please note that search-term pricing does apply when conducting SmartSELECT searches.

*
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added, *
* effective March 20, 2005. A new display format, IDERL, is now *
* available and contains the CA role and document type information. *
*

Structure search iteration limits have been increased. See HELP SLIMITS for details.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/ONLINE/UG/regprops.html>

FILE MEDLINE

FILE LAST UPDATED: 4 JAN 2006 (20060104/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 will soon be available. For details on the 2005 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate

FILE BIOSIS

FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 4 January 2006 (20060104/ED)

FILE EMBASE

FILE COVERS 1974 TO 29 Dec 2005 (20051229/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

FILE JAPIO

FILE LAST UPDATED: 02 JAN 2006 <20060102/UP>

FILE COVERS APR 1973 TO SEPTEMBER 29, 2005

<<< GRAPHIC IMAGES AVAILABLE >>>

>>> PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE
http://www.stn-international.de/stndatabases/details/ipc_reform.html <<<

FILE JICST-EPLUS

FILE COVERS 1985 TO 28 DEC 2005 (20051228/ED)

THE JICST-EPLUS FILE HAS BEEN RELOADED TO REFLECT THE 1999 CONTROLLED
TERM (/CT) THESAURUS RELOAD.

FILE USPATFULL

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 3 Jan 2006 (20060103/PD)

FILE LAST UPDATED: 3 Jan 2006 (20060103/ED)

HIGHEST GRANTED PATENT NUMBER: US6983486

HIGHEST APPLICATION PUBLICATION NUMBER: US2005289677

CA INDEXING IS CURRENT THROUGH 3 Jan 2006 (20060103/UPCA)

ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 3 Jan 2006 (20060103/PD)

REVISED CLASS FIELDS (/NCL) LAST RELOADED: Oct 2005

USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Oct 2005

>>> USPAT2 is now available. USPATFULL contains full text of the <<<
>>> original, i.e., the earliest published granted patents or <<<
>>> applications. USPAT2 contains full text of the latest US <<<
>>> publications, starting in 2001, for the inventions covered in <<<
>>> USPATFULL. A USPATFULL record contains not only the original <<<
>>> published document but also a list of any subsequent <<<
>>> publications. The publication number, patent kind code, and <<<
>>> publication date for all the US publications for an invention <<<
>>> are displayed in the PI (Patent Information) field of USPATFULL <<<
>>> records and may be searched in standard search fields, e.g., /PN, <<<

```
>>> /PK, etc. <<<
>>> USPATFULL and USPAT2 can be accessed and searched together <<<
>>> through the new cluster USPATALL. Type FILE USPATALL to <<<
>>> enter this cluster. <<<
>>> <<<
>>> Use USPATALL when searching terms such as patent assignees, <<<
>>> classifications, or claims, that may potentially change from <<<
>>> the earliest to the latest publication. <<<
```

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d que stat 121

L14 3 SEA FILE=REGISTRY ABB=ON (FORSKOLIN OR ISOFORSKOLIN OR DEACETYLFORSKOLIN)/CN
 L15 16023 SEA FILE=HCAPLUS ABB=ON (L14 OR FORSKOLIN OR ISOFORSKOLIN OR DEACETYLFORSKOLIN OR ?COLEUS?(W)?FORSKOHII)
 L16 75764 SEA FILE=HCAPLUS ABB=ON OBESITY+ALL OR WEIGHT LOSS+ALL
 L17 89 SEA FILE=HCAPLUS ABB=ON L15 AND L16
 L18 72 SEA FILE=HCAPLUS ABB=ON L17 AND (PRD<20031120 OR PD<20031120)
 L20 32 SEA FILE=HCAPLUS ABB=ON L18 AND (?PRODUC? OR ?PREP? OR ?SYNTH?)
 L21 4 SEA FILE=HCAPLUS ABB=ON L20 AND ?METHOD?

=> d ibib abs 121 1-4

L21 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN
 ACCESSION NUMBER: 2003:282110 HCAPLUS
 DOCUMENT NUMBER: 138:297697
 TITLE: Treatment **methods** based on microcompetition for a limiting GABP complex
 INVENTOR(S): Polansky, Hanan
 PATENT ASSIGNEE(S): USA
 SOURCE: U.S. Pat. Appl. Publ., 173 pp., Cont.-in-part of U.S. Ser. No. 732,360.
 CODEN: USXXCO
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 5
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003069199	A1	20030410	US 2002-219334	20020815 <--
US 2003092601	A1	20030515	US 2000-732360	20001207 <--
PRIORITY APPLN. INFO.:			US 2000-732360	A2 20001207 <--
			US 1999-169518P	P 19991207 <--
			US 2000-183184P	P 20000217 <--

AB Microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor associated with chronic disease such as **obesity**, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this discovery to present **methods** for the treatment of these chronic diseases. The **methods** are based on modifying such microcompetition, or the effect of such microcompetition on the cell. For instance, treatment may modify the cellular copy number of the foreign polynucleotide, change the rate of complex formation between GABP and either the foreign polynucleotide or the cellular GABP regulated gene, vary the expression of the cellular GABP regulated gene, or manipulate the activity of the gene **product** of the cellular GABP regulated gene. The invention also presents **methods** for treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

L21 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN
 ACCESSION NUMBER: 2002:142850 HCAPLUS
 DOCUMENT NUMBER: 136:189382
 TITLE: Bioavailable composition of natural and **synthetic** hydroxycitric acid with garcinol and anthocyanin for appetite suppression
 INVENTOR(S): Majeed, Muhammed; Badmaev, Vladimir
 PATENT ASSIGNEE(S): Sabinsa Corporation, USA

SOURCE: PCT Int. Appl., 36 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002014477	A2	20020221	WO 2001-US41748	20010817 <--
WO 2002014477	A3	20020801		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2387548	AA	20020221	CA 2001-2387548	20010817 <--
AU 2001096851	A5	20020225	AU 2001-96851	20010817 <--
AU 773081	B2	20040513		
EP 1254209	A2	20021106	EP 2001-977759	20010817 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004506657	T2	20040304	JP 2002-519605	20010817 <--
NZ 518116	A	20050624	NZ 2001-518116	20010817 <--
US 2002187943	A1	20021212	US 2002-926746	20020606 <--
PRIORITY APPLN. INFO.:			US 2000-225821P	P 20000817 <--
			WO 2001-US41748	W 20010817 <--

AB The invention relates to a composition comprising hydroxycitric acid (HCA) in combination with either one or both of garcinol and anthocyanin, and its use as a **weight-loss** therapy in animal subjects, preferably humans. The therapeutic effects for the composition observed in murine and human studies include a reduction in total body weight and body mass index, a reduction in body fat, an increase in lean body mass and content of body water, and a reduction in perceived appetite level. Another composition for use in **weight-loss** therapy is also described relating to **forskolin** in combination with either one or both of garcinol and anthocyanin. The anti-oxidant properties of garcinol are described as being enhanced in the presence of HCA and anthocyanin, and the combination of HCA, garcinol and anthocyanin is also shown to exert greater citrate lyase-inhibiting properties than either compound alone. **Methods** of obtaining HCA, garcinol or anthocyanin, or a composition containing all three compds., are described.

L21 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2000:535261 HCAPLUS

DOCUMENT NUMBER: 133:132131

TITLE: **Methods** and compositions for the differentiation of human preadipocytes into adipocytes
 Halvorsen, Yuan-Di Chang; Wilkison, William O.

INVENTOR(S):

PATENT ASSIGNEE(S): Zen-Bio, Inc., USA

SOURCE: PCT Int. Appl., 57 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000044882	A2	20000803	WO 2000-US2208	20000128 <--
WO 2000044882	A3	20010809		
W: CN, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6153432	A	20001128	US 1999-240029	19990129 <--
US 2005158706	A1	20050721	US 2005-35615	20050114 <--
PRIORITY APPLN. INFO.:			US 1999-240029	A 19990129 <--
			US 2000-585821	A3 20000601 <--

AB The present invention provides **methods** and compns. for the consistent and quant. differentiation of human preadipocytes isolated from adipose tissue into adipocytes bearing biochem., genetic, and physiol. characteristics similar to that observed in isolated primary adipocytes. The **methods** of the invention comprise incubating isolated human preadipocytes, plated at least about 25,000 cells/cm², in a medium containing, glucose, a cAMP inducer such as isobutylmethylxanthine or **(forskolin)** a glucocorticoid or glucocorticoid analog, insulin or an insulin analog and a PPAR γ agonist or a RXR agonist. Also provided are **methods** for **preparing** three dimensional biomatrices containing adipocytes differentiated by the **methods** of the invention. The compns. of the invention include human adipocytes differentiated by the **methods** of the invention, three-dimensional matrixes of adipocytes, and transfected adipocytes. The **methods** and compns. have use in the drug discovery of compds. having relevance to the disease states of diabetes, **obesity**, and cardiovascular disease and in the studies of these diseases, and in the grafting of fat tissue.

L21 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1999:753458 HCAPLUS
 DOCUMENT NUMBER: 132:1820
 TITLE: Infrared thermography for measuring real-time thermogenesis in organisms and cells
 INVENTOR(S): Lenhard, James Martin; Paulik, Mark Andrew
 PATENT ASSIGNEE(S): Glaxo Group Limited, UK
 SOURCE: PCT Int. Appl., 93 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 2
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9960630	A1	19991125	WO 1999-US10579	19990514 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,				

CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
 AU 9940774 A1 19991206 AU 1999-40774 19990514 <--
 EP 1086494 A1 20010328 EP 1999-924222 19990514 <--
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, FI
 JP 2002516398 T2 20020604 JP 2000-550152 19990514 <--
 JP 3586194 B2 20041110
 JP 2003247962 A2 20030905 JP 2003-8770 19990514 <--
 JP 2003247963 A2 20030905 JP 2003-8771 19990514 <--
 US 6881584 B1 20050419 US 1999-441493 19991117 <--
 JP 2005003681 A2 20050106 JP 2004-171280 20040609 <--
 PRIORITY APPLN. INFO.: US 1998-85736P P 19980515 <--
 JP 2000-550152 A3 19990514 <--
 WO 1999-US10579 W 19990514 <--

AB The present invention relates, in general, to thermog. and, in particular, to a **method** of using IR thermog. to monitor physiol. and mol. events that elicit a thermogenic response in animals (including humans), plants, tissues, cells and cell-free systems. The present **method** can be used for screening, identifying, and ranking drug candidates for multiple diseases, disorders and conditions. Three different inbred strains of mice, AKR/J, C57BL/6J, and SWR/J, were maintained on high and low fat diets for 14 wk before treatment with the β 3-adrenoceptor agonist, BRL37344. The heat **produced** in the intrascapular region was measured before and after 60 min treatment using IR thermog. The **obesity** prone mice, AKR/J, had a greater thermogenic response to BRL37344 when fed the higher fat diet. The **obesity** resistant mice, SWR/J, had a greater thermogenic response when fed the lower fat diet. There was little difference in the response of C57BL/6J mice on a high or low fat diet.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d'que stat 123

L14 3 SEA FILE=REGISTRY ABB=ON (FORSKOLIN OR ISOFORSKOLIN OR
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L15 16023 SEA FILE=HCAPLUS ABB=ON (L14 OR FORSKOLIN OR ISOFORSKOLIN OR
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L16 75764 SEA FILE=HCAPLUS ABB=ON OBESITY+ALL OR WEIGHT LOSS+ALL
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L20 32 SEA FILE=HCAPLUS ABB=ON L18 AND (?PRODUC? OR ?PREP? OR
?SYNTH?)
L21 4 SEA FILE=HCAPLUS ABB=ON L20 AND ?METHOD?
L22 10 SEA L21
L23 9 DUP REMOV L22 (1 DUPLICATE REMOVED)

=> d ibib abs 123 1-9

L23 ANSWER 1 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:356045 BIOSIS
DOCUMENT NUMBER: PREV200100356045
TITLE: Identification and characterization of a second
melanin-concentrating hormone receptor, MCH-2R.
AUTHOR(S): Sailer, Andreas W. [Reprint author]; Sano, Hideki; Zeng,
Zhizhen; McDonald, Terrence P.; Pan, Jie; Pong,
Sheng-Shung; Feighner, Scott D.; Tan, Carina P.; Fukami,
Takehiro; Iwaasa, Hisashi; Hreniuk, Donna L.; Morin, Nancy
R.; Sadowski, Sharon J.; Ito, Makoto; Ito, Masahiko;
Bansal, Alka; Ky, Betty; Figueroa, David J.; Jiang,
Qingping; Austin, Christopher P.; MacNeil, Douglas J.;
Ishihara, Akane; Ihara, Masaki; Kanatani, Akio; Van der
Ploeg, Lex H. T.; Howard, Andrew D.; Liu, Qingyun
CORPORATE SOURCE: Department of Metabolic Disorders (RY80Y-265), Merck
Research Laboratories, 126 East Lincoln Avenue, Rahway, NJ,
07065, USA
andreas_sailer@merck.com
SOURCE: Proceedings of the National Academy of Sciences of the
United States of America, (June 19, 2001) Vol.
98, No. 13, pp. 7564-7569. print.
CODEN: PNASA6. ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Aug 2001
Last Updated on STN: 19 Feb 2002

AB Melanin-concentrating hormone (MCH) is a 19-aa cyclic neuropeptide
originally isolated from chum salmon pituitaries. Besides its effects on
the aggregation of melanophores in fish several lines of evidence suggest
that in mammals MCH functions as a regulator of energy homeostasis.
Recently, several groups reported the identification of an orphan G
protein-coupled receptor as a receptor for MCH (MCH-1R). We hereby report
the identification of a second human MCH receptor termed MCH-2R, which
shares about 38% amino acid identity with MCH-1R. MCH-2R displayed
high-affinity MCH binding, resulting in inositol phosphate turnover and
release of intracellular calcium in mammalian cells. In contrast to
MCH-1R, MCH-2R signaling is not sensitive to pertussis toxin and MCH-2R
cannot reduce forskolin-stimulated cAMP production,
suggesting an exclusive Galphaq coupling of the MCH-2R in cell-based
systems. Northern blot and in situ hybridization analysis of human and
monkey tissue shows that expression of MCH-2R mRNA is restricted to
several regions of the brain, including the arcuate nucleus and the
ventral medial hypothalamus, areas implicated in regulation of body
weight. In addition, the human MCH-2R gene was mapped to the long arm of

chromosome 6 at band 6q16.2-16.3, a region reported to be associated with cytogenetic abnormalities of obese patients. The characterization of a second mammalian G protein-coupled receptor for MCH potentially indicates that the control of energy homeostasis in mammals by the MCH neuropeptide system may be more complex than initially anticipated.

L23 ANSWER 2 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:515542 BIOSIS
DOCUMENT NUMBER: PREV200100515542
TITLE: Modulation of the leptin-induced white adipose tissue lipolysis by nitric oxide.
AUTHOR(S): Fruhbeck, Gema [Reprint author]; Gomez-Ambrosi, Javier
CORPORATE SOURCE: Metabolic Research Laboratory, University of Navarra, 31008, Pamplona, Spain
gfruhbeck@unav.es
SOURCE: Cellular Signalling, (November, 2001) Vol. 13, No. 11, pp. 827-833. print.
CODEN: CESIEY. ISSN: 0898-6568.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Nov 2001
Last Updated on STN: 23 Feb 2002

AB The present study tested the hypothesis that nitric oxide (NO) is involved in the leptin-induced stimulation of lipolysis. The effect of intravenous (iv) administration of leptin (10, 100 and 1000 µg/kg body weight) or vehicle on serum NO concentrations and glycerol release from white adipocytes of Wistar rats was examined. One hour after injection, the three leptin doses tested increased serum NO concentrations 15.1%, 23.4% and 60.0%, respectively ($P < .001$ vs. baseline). The effect of leptin on NO concentrations was significantly dose dependent on linear trend testing ($P = .0001$). Simple linear regression analysis showed that the lipolytic rate measured was significantly correlated with serum NO concentrations ($P = .0025$; $r = .52$). In order to gain further insight into the potential underlying mechanisms, the effect of leptin on lipolysis was studied in the setting of nitric oxide **synthase** (NOS) inhibition or acute ganglionic blockade. The stimulatory effect of leptin on lipolysis was significantly decreased ($P < .05$) under NOS inhibition. On the contrary, the leptin-induced lipolysis was unaltered in pharmacologically induced ganglionic blockade. The lack of effect on isoproterenol-, **forskolin**- and dibutyryl-cyclic AMP-stimulated lipolysis suggests that leptin does not interfere with the signal transduction pathway at the beta-adrenergic receptor, the adenylate cyclase and the protein kinase A levels. These findings suggest that NO is a potential regulator of leptin-induced lipolysis.

L23 ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:428724 BIOSIS
DOCUMENT NUMBER: PREV200100428724
TITLE: Identification of potent and selective neuropeptide Y Y1 receptor agonists with orexigenic activity in vivo.
AUTHOR(S): Mullins, Deborra [Reprint author]; Kirby, Dean; Hwa, Joyce; Guzzi, Mario; Rivier, Jean; Parker, Eric
CORPORATE SOURCE: Schering-Plough Research Institute, 2015 Galloping Hill Rd., Kenilworth, NJ, 07033, USA
deborra.mullins@spcorp.com
SOURCE: Molecular Pharmacology, (September, 2001) Vol. 60, No. 3, pp. 534-540. print.
CODEN: MOPMA3. ISSN: 0026-895X.
DOCUMENT TYPE: Article
LANGUAGE: English

ENTRY DATE: Entered STN: 12 Sep 2001

Last Updated on STN: 22 Feb 2002

AB Neuropeptide Y (NPY) binds to a family of G-protein coupled receptors termed Y1, Y2, Y3, Y4, Y5, and y6. The use of various receptor subtype-selective agonists and antagonists has facilitated identification of the receptor subtypes responsible for mediating many of the biological effects of NPY. For example, the potent orexigenic activity of NPY is believed to be mediated by both the Y1 and Y5 receptor subtypes. Several selective Y5 receptor agonists that stimulate food intake in rodents are available, but no selective Y1 receptor agonist has been reported. We have identified several NPY analogs that bind the NPY Y1 receptor with high affinity and exhibit full agonist activity, measured as inhibition of **forskolin-stimulated cAMP production** in cells expressing the cloned NPY Y1 receptor. (D-Arg25)-NPY, (D-His26)-NPY, Des-AA10-17(Cys7,21,Pro34)-NPY, Des-AA11-18(Cys7,21,D-Lys9(Ac))-NPY, Des-AA11-18(Cys7,21,D-Lys9(Ac),Pro34)-NPY, Des-AA11-18(Cys7,21,D-Lys9(Ac),D-His26)-NPY and Des-AA11-18(Cys7,21,D-Lys9(Ac),D-His26,Pro34)-NPY bind the NPY Y1 receptor with K_i values of 0.9 ± 0.2 , 2.0 ± 0.3 , 0.2 ± 0.05 , 0.7 ± 0.1 , 0.2 ± 0.01 , 2.2 ± 0.3 , and 1.2 ± 0.3 nM, respectively, and inhibit **forskolin-stimulated cAMP production** with EC_{50} values of 0.2 ± 0.02 , 0.5 ± 0.04 , 0.3 ± 0.03 , 0.5 ± 0.05 , 0.4 ± 0.16 , 5.3 ± 0.32 , and 5.1 ± 0.97 nM, respectively. These peptides are highly selective for the NPY Y1 receptor relative to the NPY Y2, Y4, and Y5 receptors. (D-Arg25)-NPY, (D-His26)-NPY and Des-AA11-18(Cys7,21,D-Lys9(Ac),D-His26,Pro34)-NPY stimulate food intake dose-responsively in Long-Evans rats for at least 4 h after intracerebroventricular administration. Although the involvement of Y1 receptors in several physiological activities, such as vasoconstriction and anxiolysis, remains to be investigated, adequate tools are now available.

L23 ANSWER 4 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:360002 BIOSIS
DOCUMENT NUMBER: PREV199900360002
TITLE: Divergent effects of weight reduction and oral
anticonception treatment on adrenergic lipolysis regulation
in obese women with the polycystic ovary syndrome.
AUTHOR(S): Wahrenberg, Hans [Reprint author]; Ek, Ingvar;
Reynisdottir, Signy; Carlstrom, Kjell; Bergqvist, Agneta;
Arner, Peter
CORPORATE SOURCE: Center of Metabolism and Endocrinology, Department of
Medicine M63, Huddinge Hospital, Karolinska Institute,
S-141 86, Huddinge, Sweden
SOURCE: Journal of Clinical Endocrinology and Metabolism, (
June, 1999) Vol. 84, No. 6, pp. 2182-2187. print.
CODEN: JCEMAZ. ISSN: 0021-972X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Sep 1999
Last Updated on STN: 2 Sep 1999

AB The influence of weight reduction and female sex hormones on the regulation of lipolysis was investigated in isolated abdominal sc adipocytes from 20 obese hyperandrogenic women with polycystic ovary syndrome (PCOS). Nine PCOS women were reinvestigated after 8-12 weeks of weight reduction therapy (WR) with a very low calorie diet, inducing a mean loss of 8 ± 3 kg, and 8 PCOS women were reinvestigated after 12 weeks of treatment with combined oral contraceptives (OC), containing ethinyl estradiol and norethisterone; the remaining 3 subjects were drop-outs. Both WR and OC normalized hyperandrogenicity. WR caused a 50% reduction of basal lipolysis rate and a 5- to 7-fold increased noradrenaline and terbutaline sensitivity ($P < 0.02$); the latter could be

ascribed to a 2-fold increased beta2adrenoceptor density ($P < 0.02$) as determined with radioligand binding. There was no change with regard to dobutamine (beta1-adrenoceptor sensitivity) or clonidine, (alpha-adrenoceptor sensitivity) or to beta1-adrenoceptor density. OC treatment did not influence the basal lipolysis rate or beta2- or alpha2-adrenoceptor sensitivity, but lowered the beta1-adrenoceptor sensitivity 7-fold ($P 0.03$) without a reduction in beta1-adrenoceptor density. The OC treatment effect was not observed when **forskolin** and dibutyryl cAMP, acting on adenylate cyclase or protein kinase A, respectively, were used, suggesting a partial uncoupling of beta1-adrenoceptors. WR therapy, but not OC therapy, caused, in addition to changes in lipolysis function, improved in vivo insulin sensitivity and lower plasma noradrenaline levels. These findings suggest that factors other than hyperandrogenicity modulate lipolysis regulation in obese subjects with PCOS. Disturbances in sympathetic pathways could be of pathogenic importance.

L23 ANSWER 5 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
 ACCESSION NUMBER: 1998:315857 BIOSIS
 DOCUMENT NUMBER: PREV199800315857
 TITLE: Ability of various bombesin receptor agonists and

antagonists to alter intracellular signaling of the human orphan receptor BRS-3.

AUTHOR(S): Ryan, Richard R.; Weber, H. Christian; Hou, Wei; Sainz, Eduardo; Mantey, Samuel A.; Battey, James F.; Coy, David H.; Jensen, Robert T. [Reprint author]

CORPORATE SOURCE: NIH/NIDDK/DBD, Build. 10, Room 9C-103, 10 Center Dr., MSC 1804, Bethesda, MD 20892-1804, USA

SOURCE: Journal of Biological Chemistry, (May 29, 1998)
 Vol. 273, No. 22, pp. 13613-13624. print.
 CODEN: JBCHA3. ISSN: 0021-9258.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 22 Jul 1998
 Last Updated on STN: 22 Jul 1998

AB Bombesin (Bn) receptor subtype 3 (BRS-3) is an orphan receptor that is a predicted member of the hepta-helical G-protein receptor family and so named because it shares a 50% amino acid homology with receptors for the mammalian bombesin-like peptides neuromedin B (NMB) and gastrin-releasing peptide. In a recent targeted disruption study, in which BRS-3-deficient mice were generated, the mice developed **obesity**, diabetes, and hypertension. To date, BRS-3's natural ligand remains unknown, its pharmacology unclear, and cellular basis of action undetermined. Furthermore, there are few tissues or cell lines found that express sufficient levels of BRS-3 protein for study. To define the intracellular signaling properties of BRS-3, we examined the ability of (D-Phe6,beta-Ala11,Phe13,Nle14)Bn-(6-14), a newly discovered peptide with high affinity for BRS-3, and various Bn receptor agonists and antagonists to alter cellular function in hBRS-3-transfected BALB 3T3 cells and hBRS-3-transfected NCI-H1299 non-small cell lung cancer cells, which natively express very low levels of hBRS-3. This ligand stimulated a 4-9-fold increase in (3H)inositol phosphate formation in both cell lines under conditions where it caused no stimulation in untransfected cells and also stimulated an increase in (3H)IP1 (3H)IP2, and (3H)IP3. The elevation of (3H)IP was concentration-dependent, with an EC50 of 20-35 nM in both cell lines. (D-Phe6,beta-Ala11,Phe13,Nle14)Bn-(6-14) stimulated a 2-3-fold increase in (Ca2+)i, a 3-fold increase in tyrosine phosphorylation of p125FAK with an EC50 of 0.2-0.7 nM, but failed to either stimulate increases in cyclic AMP or inhibit **forskolin**-stimulated increases. None of nine naturally occurring Bn peptides or

three **synthetic** Bn analogues reported to activate hBRS-3 did so with high affinity. No high affinity Bn receptor antagonists had high affinity for the hBRS-3 receptor, although two low affinity antagonists for gastrin-releasing peptide and NMB receptors, (D-Arg1,DTrp7,9,Leu11)substance P and (D-Pro4,D-Trp7,9,10)substance P-(4-11), inhibited hBRS-3 receptor activation. The NMB receptor-specific antagonist D-Nal,Cys,Tyr,D-Trp,Lys,Val, Cys,Nal-NH2 inhibited hBRS-3 receptor activation in a competitive fashion ($K_i = 0.5 \mu\text{M}$). Stimulation of p125FAK tyrosine phosphorylation by hBRS-3 activation was not inhibited by the protein kinase C inhibitor, GF109203X, or thapsigargin, alone or in combination. These results show that hBRS-3 receptor activation increases phospholipase C activity, which causes generation of inositol phosphates and changes in $(\text{Ca}^{2+})_i$ and is also coupled to tyrosine kinase activation, but is not coupled to adenylate cyclase activation or inhibition. hBRS-3 receptor activation results in tyrosine phosphorylation of p125FAK, and it is not dependent on activation of either limb of the phospholipase C cascade. Although the natural ligand is not a known bombesin-related peptide, the availability of (D-Phe6,beta-Ala11,Phe13,Nle14)Bn-(6-14), which functions as a high affinity agonist in conjunction with hBRS-3-transfected cell lines and the recognition of three classes of receptor antagonists including one with affinity of $0.5 \mu\text{M}$ should provide important tools to assist in the identification of its natural ligand, the development of more potent selective receptor antagonists and agonists, and further exploration of the signaling properties of the hBRS-3 receptor.

L23 ANSWER 6 OF 9 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1998055898 EMBASE
TITLE: Effects of leptin on insulin secretion from isolated rat pancreatic islets.
AUTHOR: Ookuma M.; Ookuma K.; York D.A.
CORPORATE SOURCE: Dr. D.A. York, Pennington Biomed. Research Center, 6400 Perkins Rd., Baton Rouge, LA 70808, United States.
yorkda@mhs.pbrc.edu
SOURCE: Diabetes, (1998) Vol. 47, No. 2, pp. 219-223.
Refs: 42
ISSN: 0012-1797 CODEN: DIAEAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 19980416
Last Updated on STN: 19980416

AB Leptin is a hormone secreted by adipocytes as a peripheral metabolic signal for the central regulation of energy homeostasis or the **reproductive** system. Recent studies demonstrated that leptin receptor mRNA is expressed in pancreatic islets of rodents and that leptin at relatively high doses inhibits glucose-induced insulin secretion from rat islets. However, the physiological mechanism of leptin on insulin secretion has not been identified. In this study, we report that leptin inhibits glucose-induced insulin secretion at lower concentrations ranging from 25 to 50 ng/ml using a static incubation **method**. A perifusion study revealed that leptin (50 ng/ml) affected the second phase of insulin secretion but not the first phase. Leptin did not affect insulin secretion stimulated by glibenclamide (1 and 5 $\mu\text{mol/l}$) or **forskolin** (1 $\mu\text{mol/l}$). Leptin (50 ng/ml) significantly inhibited insulin secretion induced by the phorbol ester phorbol

12-myristate 13- acetate (TPA) in the presence of Ca²⁺ but not in the absence of Ca²⁺ . Because TPA is known to activate protein kinase C (PKC), these present results suggest that leptin, at a physiological concentration, suppresses the second phase of insulin secretion by reducing activity of the Ca²⁺- dependent PKC isoform.

L23 ANSWER 7 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1993:477013 BIOSIS
DOCUMENT NUMBER: PREV199396110613
TITLE: Growth hormone secretion is differently affected in
genetically obese male and female rats.
AUTHOR(S): Cocchi, Daniela [Reprint author]; Parenti, Marco; Cattaneo,
Lorena; Colonna, V. De Gennaro; Zocchetti, Andrea; Mueller,
Eugenio E.
CORPORATE SOURCE: Dep. Pharmacol., University Milan Sch. Med., Via
Vanvitelli, 32, I-20129 Milan, Italy
SOURCE: Neuroendocrinology, (1993) Vol. 57, No. 5, pp.
928-934.
CODEN: NUNDAJ. ISSN: 0028-3835.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Oct 1993
Last Updated on STN: 23 Oct 1993
AB Growth hormone (GH) secretion is markedly blunted in **obesity**.
Reportedly, genetically obese Zucker rats show a reduced GH secretion due
to an impaired function of hypothalamic neurons **producing** the
GH-releasing hormone (GHRH). The aim of this work was: (1) to compare the
in vitro GH responsiveness to GHRH in genetically obese female versus male
Zucker rats and, (2) to evaluate the function of hypothalamic GHRH and
somatostatin and of pituitary receptors for these neurohormones as
assessed by the effectiveness of GHRH and somatostatin on adenylate
cyclase (AC) activity. Baseline GH secretion of pituitaries obtained from
male and female obese rats was not different and similar to that present
in lean counterparts. Stimulation with 10⁻⁷ M GHRH elicited a
significantly lower GH secretion from the pituitaries of lean and obese
female rats. In these pituitaries of obese male rats, but induced a
similar GH secretion from the pituitaries, GH concentrations was similar
in obese versus lean male and female rats. A sex-related difference was
also evidenced when plasma concentrations of somatomedin C (IGF-I) were
evaluated. Obese male rats had lower IGF-I concentrations than lean
counterparts, while this was not the case for obese versus lean female
rats. Evaluation of AC activity following GHRH disclosed a lower
activation in obese than in lean male rats, whereas in the females the
enzyme activation was higher in obese than in lean animals. Conversely,
the inhibitory effect of somatostatin on **forskolin**-stimulated AC
was similar in pituitary membranes of obese and lean rats of both sexes.
Determination of GHRH mRNA in the hypothalamus of obese rats showed that
it was significantly reduced in male but not in female obese rats versus
lean counterparts. In contrast, somatostatin mRNA concentrations were
unchanged in the hypothalamus of obese rats of both sexes. Overall these
data suggest that despite an in vivo reduced GH secretion, the
hypothalamo-pituitary GH regulatory system is more preserved in female
than in male obese rats.

L23 ANSWER 8 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 1
ACCESSION NUMBER: 1993:225988 BIOSIS
DOCUMENT NUMBER: PREV199395117163
TITLE: Effects of **obesity** and hypertension on
ventricular myocytes: Comparison of cells from adult

SHHF/Mcc-cp and JCR:LA-cp rats.
AUTHOR(S): Hohl, Charlene M.; Hu, Bo; Fertel, Richard H.; Russell, James C.; McCune, Sylvia A.; Altschuld, Ruth A. [Reprint author]
CORPORATE SOURCE: Dep. Med. Biochem., Ohio State Univ., 333 Hamilton Hall, 1645 Neil Ave., Columbus, OH 43210-1218, USA
SOURCE: Cardiovascular Research, (1993) Vol. 27, No. 2, pp. 238-242.
CODEN: CVREAU. ISSN: 0008-6363.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 7 May 1993
Last Updated on STN: 8 May 1993
AB Objective: The aim was to compare beta adrenergic receptors, cAMP **production**, and Ca-2+ accumulation by the sarcoplasmic reticulum in ventricular cardiomyocytes from female SHHF/Mcc-cp and JCR:LA-cp rats. Whereas rats from both strains exhibit gross **obesity** when the animals are homozygous for the recessive "corpulent" gene, the SHHF rats, which are hypertensive, all develop heart failure during their second year of life. The normotensive JCR:LA-cp animals do not. **Methods:** beta Adrenergic receptor number, ligand affinity, isoprenaline and **forskolin** stimulated cyclic AMP **production**, and ATP dependent, phosphate supported 45Ca-2+ uptake by the sarcoplasmic reticulum were compared in ventricular cardiomyocytes isolated from 6 month old obese female SHHF/Mcc-cp and obese and lean female JCR:LA-cp rats. Results: B-max and K-d for (-)-(125iodo)-cyanopindolol (125ICYP) binding were each apprx 50% lower in SHHF/Mcc-cp v JCR:LA-cp myocytes. Cyclic AMP **production** in response to isoprenaline, isoprenaline plus isobutylmethylxanthine (IBMX), and **forskolin** plus IBMX was also significantly depressed in the SHHF/Mcc-cp cells. In addition, sarcoplasmic reticular 45Ca-2+ uptake by SHHF/Mcc-cp cells was 35% lower than in lean or obese JCR:LA-cp myocytes. Isoprenaline stimulated cAMP **production** and sarcoplasmic reticular Ca-2+ uptake by the lean JCR:LA-cp cells were comparable to that described previously for myocytes from normal Sprague-Dawley rats. By contrast, B-max and K-d for 125ICYP binding by the JCR myocytes differed substantially from previously described results for normal Sprague-Dawley rats, whereas values for the SHHF cells did not. Conclusions: Declines in Ca sequestration by the sarcoplasmic reticulum of ventricular cardiomyocytes from obese, hypertensive SHHF rats are not related to their **obesity**. However, **obesity** may contribute to the decline in cAMP **production**. This may account, in part, for the exacerbation by **obesity** of cardiac dysfunction in essential hypertension.

L23 ANSWER 9 OF 9 JAPIO (C) 2006 JPO on STN

ACCESSION NUMBER: 2001-206893 JAPIO

TITLE: NEW α -AMYLASE INHIBITORY ACTIVE SUBSTANCE,
METHOD FOR PRODUCING THE SAME AND ITS USE

INVENTOR: KAWAKAMI TOSHIOKI; UCHIDA AYUMI

PATENT ASSIGNEE(S): SANPO KK
HEALTH WAY:KK

PATENT INFORMATION:

PATENT NO	KIND	DATE	ERA	MAIN IPC
JP 2001206893	A	20010731	Heisei	C07G017-00

APPLICATION INFORMATION

STN FORMAT: JP 2000-346380 20001114

ORIGINAL: JP2000346380 Heisei
PRIORITY APPLN. INFO.: JP 1999-327361, 19991117
SOURCE: PATENT ABSTRACTS OF JAPAN (CD-ROM), Unexamined
Applications, Vol. 2001

AN 2001-206893 JAPIO

AB PROBLEM TO BE SOLVED: To obtain an excellent food, beverage and medicine capable of preventing, treating or improving diabetic mellitus or **obesity**, or a component effective for the above purposes.
SOLUTION: This new α -amylase inhibitory active substance totally different from **forskolin** which is conventionally considered to be the main physiologically active substance of *Coleus forskohli*, and containing an alcohol soluble compound exhibiting such activity not observed in **forskolin** is isolated from the *Coleus forskohli*. The α -amylase inhibitory substance has a suppressing activity of the elevation of blood sugar, and preferably α -glucosidase inhibitory activity and/or lipase inhibitory activity, is useful for the prevention, treatment, improvement, etc., of diabetes mellitus and **obesity**, and is expected to be used as a medicine, food or beverage for such purpose.

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=> d que stat 126

L14 3 SEA FILE=REGISTRY ABB=ON (FORSKOLIN OR ISOFORSKOLIN OR DEACETYLFORSKOLIN)/CN

L15 16023 SEA FILE=HCAPLUS ABB=ON (L14 OR FORSKOLIN OR ISOFORSKOLIN OR DEACETYLFORSKOLIN OR ?COLEUS?(W)?FORSKOHII)

L16 75764 SEA FILE=HCAPLUS ABB=ON OBESITY+ALL OR WEIGHT LOSS+ALL

L17 89 SEA FILE=HCAPLUS ABB=ON L15 AND L16

L18 72 SEA FILE=HCAPLUS ABB=ON L17 AND (PRD<20031120 OR PD<20031120)

L20 32 SEA FILE=HCAPLUS ABB=ON L18 AND (?PRODUC? OR ?PREP? OR ?SYNTH?)

L24 889 SEA FILE=USPATFULL ABB=ON L20 AND ?METHOD?

L25 876 SEA FILE=USPATFULL ABB=ON L24 AND ?METHOD?(L)?PREP?

L26 19 SEA FILE=USPATFULL ABB=ON L25 AND ?LEAN?(W)?BODY?(W)?MASS?

=> d ibib abs 126 1-19

L26 ANSWER 1 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2005:203402 USPATFULL

TITLE: Compositions and **methods** for glycogen **synthesis**

INVENTOR(S): Lee, Steve S., Sandy, UT, UNITED STATES
 Hynson, Richard B., Missoula, MT, UNITED STATES
 Ruby, Brent C., Missoula, MT, UNITED STATES
 Gaskill, Steven E., Missoula, MT, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005176827	A1	20050811
APPLICATION INFO.:	US 2004-926849	A1	20040826 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2003-434444, filed on 7 May 2003, PENDING		

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2002-379839P	20020510 (60)	<--
	US 2003-498717P	20030828 (60)	<--
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	PATE PIERCE & BAIRD, 215 SOUTH STATE STREET, SUITE 550, PARKSIDE TOWER, SALT LAKE CITY, UT, 84111, US		
NUMBER OF CLAIMS:	64		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Page(s)		
LINE COUNT:	2907		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A composition of bio-active compounds and **methods** for facilitating and supporting the metabolism and transport of glucose and carbohydrates into muscle cells, promoting muscle function and growth, promoting glycogen **synthesis**, enhancing glucose disposal, stimulating pancreatic beta cells, promoting metabolic recovery, promoting muscle recovery, promoting **lean body mass**, and promoting fat burning. Preferably, the composition of bio-active compounds includes a combination of 4-hydroxyisoleucine with at least one amino acid selected from the group consisting of arginine, aspartate, threonine, serine, glutamate, proline, glycine, alanine, cysteine, valine, methionine, isoleucine, leucine, tryptophan, phenylalanine, ornithine, lysine, histidine, gamma-amino butyrate and tyrosine. In one presently preferred embodiment of the present invention, the combination is derived, isolated, and/or extracted from fenugreek seeds. **Methods** for using a novel composition of

bio-active compounds from fenugreek seed for facilitating and supporting the metabolism and transport of glucose and carbohydrates into muscle cells, promoting muscle function and growth, promoting glycogen **synthesis**, enhancing glucose disposal, stimulating pancreatic beta cells, promoting metabolic recovery, promoting muscle recovery, promoting **lean body mass**, and promoting fat burning are also disclosed, wherein **methods** comprise the steps of: (1) providing an effective amount of a composition of bio-active compounds derived, isolated, and/or extracted from fenugreek seeds; and (2) administering the composition to a human or animal.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 2 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2005:75107 USPATFULL

TITLE: Regulation of human serotonin-like g protein-coupled receptor

INVENTOR(S): Smolyar, Alex, Brookline, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005064404	A1	20050324
APPLICATION INFO.:	US 2003-470150	A1	20031217 (10)
	WO 2002-EP540		20020121

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-264071P	20010126 (60)	<--
	US 2001-324054P	20010924 (60)	<--
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001		
NUMBER OF CLAIMS:	45		
EXEMPLARY CLAIM:	CLM-01-71		
NUMBER OF DRAWINGS:	16 Drawing Page(s)		
LINE COUNT:	4366		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Reagents which regulate human serotonin-like G protein-coupled receptor (5-HT-like GPCR) and reagents which bind to human 5-HT-like GPCR gene **products** can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, COPD, cardiovascular disorders, cancer, urinary disorders, **obesity**, diabetes, CNS disorders, asthma, and hematological disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 3 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2004:260183 USPATFULL

TITLE: **Method** of enhanced regional body fat reduction

INVENTOR(S): Hopkins, Kevin J., Washington, NJ, UNITED STATES
Dente, Gerard, Cedar Grove, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004202677	A1	20041014
APPLICATION INFO.:	US 2004-822248	A1	20040408 (10)

NUMBER	DATE
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PRIORITY INFORMATION: US 2003-461435P, 20030410 (60) <--
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MELVIN K. SILVERMAN, 500 WEST CYPRESS CREEK ROAD, SUITE
500, FT. LAUDERDALE, FL, 33309
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 469

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A **method** of enhancing regional body fat reduction is disclosed. The **method** includes topically applying a fat reduction topical composition on a region of a human body where a regional fat reduction is desired for a treatment period, and administering an amount of a fat reduction oral composition daily during the treatment period. The topical composition includes hydroglycolic fluid extract of *Palmaria palmata*, *Laminaria digitata* extract, mannitol, and a pharmaceutically acceptable carrier. The oral composition includes synephrine, methylxanthine, chromium, a herbal combination having diuretic effect and a pharmaceutically acceptable carrier.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 4 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2004:190687 USPATFULL
TITLE: Novel composition for the treatment of **obesity**
and effective fat loss promotion
INVENTOR(S): Ramazanov, Arthur, Warwick, NY, UNITED STATES
Ramazanov, Zakir, Warwick, NY, UNITED STATES
PATENT ASSIGNEE(S): National Bioscience Corporation (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004147460	A1	20040729
APPLICATION INFO.:	US 2003-660256	A1	20030911 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-438113P	20030106 (60) <--
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	DARBY & DARBY P.C., Post Office Box 5257, New York, NY, 10150-5257	
NUMBER OF CLAIMS:	11	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	3 Drawing Page(s)	
LINE COUNT:	986	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention encompasses pharmaceutical compositions for the treatment of **obesity**. These compositions comprise dihydroquercetins (dihydroquercetin 3-rhamnoside and its aglycon dihydroquercetin) and the triterpene saponins known as aralosides or elatosides. The compositions of the present invention effectively promote total **weight loss** and body fat mass loss. Therefore, the present invention is also directed to **methods** for treating **obesity**, reducing total weight, and reducing body fat mass by administering the compositions of the invention. The invention also embraces **methods** for disrupting the perilipin shell of lipid droplets and stimulating the activity of hormone-sensitive lipase.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 5 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2004:101687 USPATFULL

TITLE: Compositions and **methods** for promoting **weight loss**, thermogenesis, appetite suppression, lean muscle mass, increasing metabolism and boosting energy levels, and use as a dietary supplement in mammals

INVENTOR(S): Chinery, Robert, Spring Lake, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004077556	A1	20040422
APPLICATION INFO.:	US 2003-419974	A1	20030422 (10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2002-374505P	20020422 (60)	<--
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	CATALINA + ASSOCIATES, LLC, 167 AVENUE AT THE COMMON, SUITE 9, SECOND FLOOR, SHREWSBURY, NJ, 07702		
NUMBER OF CLAIMS:	5929		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	1 Drawing Page(s)		
LINE COUNT:	30974		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention is both a composition and a **method** for promoting **weight loss** in mammals, for promoting thermogenesis in mammals, for increasing metabolism and boosting energy levels in mammals, promoting appetite suppression in mammals, for promoting lean muscle mass in mammals and for a diet supplement. The primary mechanism of action for the invention is that it increases norepinephrine levels, which promotes a rise in metabolism, thus leading to more calories burned and more energy expended primarily through the burning or metabolism of adipose tissue (fat) through lipolysis, without the destruction or metabolism of muscle tissue.

The present invention relates to a nutritional supplement composition, and the **methods** for the administration thereof, comprising of (1) an effective amount of epigallocatechin gallate (EGCG), the chemical name of which has been described as ((2R,3R)-2-(3,4,5-Trihydroxyphenyl)-3,4-dihydro-1[2H]-benzopyran-3,5,7-triol 3-(3,4,5-trihydroxybenzoate), and (2) various other substances (singly or in any combination thereof), which either (a) inhibit cyclic adenosine monophosphate (cAMP) phosphodiesterase, (b) stimulate lipolysis, (c) stimulate thermogenesis (i.e., increase metabolism) (d) and/or increase norepinephrine levels, or (e) any combination thereof.

In a preferred embodiment, the basic invention is a composition, and a **method** for the administration thereof, comprising effective amounts of epigallocatechin gallate, caffeine, and l-tyrosine.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 6 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2003:300776 USPATFULL

TITLE: Peptides and **methods** for the control of

obesity
INVENTOR(S): Haskell-Luevano, Carrie, Archer, FL, UNITED STATES
Holder, Jerry R., Gainesville, FL, UNITED STATES
PATENT ASSIGNEE(S): University of Florida (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003212002	A1	20031113	<--
APPLICATION INFO.:	US 2002-139624	A1	20020507	(10)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	Dennis P. Clarke, Miles & Stockbridge, Suite 500, 1751 Pinnacle Drive, McLean, VA, 22102-3833			
NUMBER OF CLAIMS:	10			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	4 Drawing Page(s)			
LINE COUNT:	681			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a peptide derivative having the formula:

X.sup.1--Z--Q-arg-trp-NH.sub.2

Wherein: X.sup.1 is an acyl group,

Z is amino-2-naphthyl-carboxylic acid or histidine,

Q is (D)phenylalanine or p-iodo-(D)phenylalanine, or a pharmacologically acceptable salt, complex or derivative thereof, the peptide derivative having melanocortin-4 receptor agonist activity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 7 OF 19 USPATFULL on STN
ACCESSION NUMBER: 2003:244447 USPATFULL
TITLE: Regulation of human map kinase phosphatase-like enzyme
INVENTOR(S): Liou, Jiing-Ren, Belmont, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003170856	A1	20030911	<--
APPLICATION INFO.:	US 2003-363676	A1	20030424	(10)
	WO 2001-EP9848		20010827	
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001			
NUMBER OF CLAIMS:	71			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	13 Drawing Page(s)			
LINE COUNT:	3736			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Reagents which regulate human MAP kinase phosphatase-like enzyme and reagents which bind to human MAP kinase phosphatase-like enzyme gene **products** can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, allergies including asthma, CNS disorders, diabetes, **obesity**, chronic obstructive pulmonary disease, cancer, and cardiovascular diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 8 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2003:213711 USPATFULL

TITLE: Regulation of human 11 beta-hydroxysteroid
dehydrogenase 1-like enzyme

INVENTOR(S): Ramakrishnan, Shyam, Brighton, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003148349	A1	20030807	<--
APPLICATION INFO.:	US 2003-312831	A1	20030103	(10)
	WO 2001-EP7632		20010704	
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100,			
	WASHINGTON, DC, 20001			
NUMBER OF CLAIMS:	71			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	7 Drawing Page(s)			
LINE COUNT:	2445			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Reagents which regulate human 11 beta-hydroxysteroid dehydrogenase 1-like enzyme and reagents which bind to human 11 beta-hydroxysteroid dehydrogenase 1-like enzyme gene **products** can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to CNS disorders, osteoporosis, liver disease, **obesity**, blood pressure or fetal development abnormalities, and diabetes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 9 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2003:165409 USPATFULL

TITLE: **Methods** and reagents for using mammalian
melanocortin receptor antagonists to treat cachexia

INVENTOR(S): Marks, Daniel L., Portland, OR, UNITED STATES

Cone, Roger D., Oregon City, OR, UNITED STATES

PATENT ASSIGNEE(S): Oregon Health and Sciences University, a non-profit
organization, Portland, OR (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003113263	A1	20030619	<--
APPLICATION INFO.:	US 2002-74754	A1	20020213	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-268357P	20010213	(60) <--
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606		
NUMBER OF CLAIMS:	15		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	15 Drawing Page(s)		
LINE COUNT:	1891		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides recombinant expression constructs comprising nucleic acid encoding mammalian melanocortin receptors, in particular MC-4 melanocortin receptor, and mammalian cells into which

said recombinant expression constructs have been introduced that express functional mammalian MC-4 melanocortin receptors. The invention particularly provides such genetically engineered cells expressing the human MC4-R melanocortin receptor for screening compounds for receptor agonist and antagonist activity. The invention also provides screening **methods** using genetically engineered cells expressing the human MC-4 melanocortin receptor to specifically detect and identify agonists and antagonists for this melanocortin receptor. Such screening **methods** are provided identifying compounds with MC-4 melanocortin receptor antagonist activity having the capacity to influence or modify metabolism and feeding behavior, particularly pathological feeding behavior such as illness-induced cachexia.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 10 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2003:152692 USPATFULL
TITLE: Diagnosis **methods** based on microcompetition
for a limiting GABP complex
INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003104358	A1	20030605	<--
APPLICATION INFO.:	US 2002-219649	A1	20020815	(10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623			
NUMBER OF CLAIMS:	32			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	28 Drawing Page(s)			
LINE COUNT:	14430			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Microcompetition for GABP between a foreign polynucleotide and cellular GABP regulated genes is a risk factor associated with many chronic diseases such as **obesity**, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present assays for the diagnosis of these chronic diseases. The assays are based on measuring the cellular copy number of the foreign polynucleotide, measuring the rate of complex formation between GABP and either the foreign polynucleotide, or a cellular GABP regulated gene, identifying modified expression of a cellular GABP regulated gene, or identifying modified activity of the gene **product** of a GABP regulated gene. The invention also presents other foreign polynucleotide-type assays.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 11 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2003:134514 USPATFULL
TITLE: Microcompetition and human disease
INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003092601	A1	20030515	<--
APPLICATION INFO.:	US 2000-732360	A1	20001207	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1999-169518P	19991207 (60)	<--
	US 2000-183184P	20000217 (60)	<--
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Brown, Pinnisi and Michaels, P.C., 400 M&T Bank Building-118 North Tioga Street, Ithaca, NY, 14850-4343		
NUMBER OF CLAIMS:	50		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	8 Drawing Page(s)		
LINE COUNT:	7921		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Cellular microcompetition for the transcription factor human GA binding protein (GABP) is a risk factor associated with **obesity** and **obesity**-related diseases such as osteoarthritis, atherosclerosis, obstructive sleep apnea, various cancers, and periodontitis. The invention uses this novel discovery to develop assays which determine the level of microcompetition in a cell. Other assays developed from the knowledge that microcompetition is occurring in cells are also disclosed. This novel discovery led to the development of assays which can determine the level of microcompetition in a cell and to select compounds to target this microcompetition syndrome. In addition, **methods** to treat a patient for microcompetition based disease are taught.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 12 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2003:106204 USPATFULL
TITLE: Regulation of human galanin receptor-like g protein coupled receptor
INVENTOR(S): Ramakrishnan, Shyam, Brighton, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003073115	A1	20030417	<--
APPLICATION INFO.:	US 2002-221737	A1	20020916 (10)	
	WO 2001-EP2925		20010315	
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001			
NUMBER OF CLAIMS:	18			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	2 Drawing Page(s)			
LINE COUNT:	3657			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Reagents which regulate human galanin receptor-like GPCR and reagents which bind to human galanin receptor-like gene **products** can be used to regulate the effect of galanin for therapeutic purposes. Treatment of pathophysiological disorders such as eating disorders, including **obesity**, diabetes, cardiovascular disease, asthma, pain, depression, ischemia, Alzheimer's disease, sleep disorders, migraine, anxiety, and **reproductive** disorders can be treated. Processes such as cognition, analgesia, sensory processing (olfactory, visual), processing or visceral information, motor coordination, modulation of dopaminergic activity, and neuroendocrine function can be modulated.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 13 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2003:100088 USPATFULL
TITLE: Treatment **methods** based on microcompetition
for a limiting GABP complex
INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003069199	A1	20030410	<--
APPLICATION INFO.:	US 2002-219334	A1	20020815	(10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623			
NUMBER OF CLAIMS:	26			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	28 Drawing Page(s)			
LINE COUNT:	14837			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor associated with chronic disease such as **obesity**, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present **methods** for the treatment of these chronic diseases. The **methods** are based on modifying such microcompetition, or the effect of such microcompetition on the cell. For instance, treatment may modify the cellular copy number of the foreign polynucleotide, change the rate of complex formation between GABP and either the foreign polynucleotide or the cellular GABP regulated gene, vary the expression of the cellular GABP regulated gene, or manipulate the activity of the gene **product** of the cellular GABP regulated gene. The invention also presents **methods** for treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 14 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2003:99511 USPATFULL
TITLE: Drug discovery assays based on microcompetition for a
limiting GABP complex
INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003068616	A1	20030410	<--
APPLICATION INFO.:	US 2002-223050	A1	20020814	(10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623			
NUMBER OF CLAIMS:	55			
EXEMPLARY CLAIM:	1			

NUMBER OF DRAWINGS: 28 Drawing Page(s)

LINE COUNT: 14981

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A recent discovery showed that microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor for some of the major chronic diseases, such as **obesity**, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present assays for screening compounds based on their effectiveness in modulating such microcompetition, or the effects of such microcompetition on the cell. The selected compounds can be used in treatment of these chronic diseases. The invention also presents assays for screening compounds that can be used in treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 15 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2002:336907 USPATFULL

TITLE: Materials and **methods** for the treatment or prevention of **obesity**

INVENTOR(S): Zemel, Michael B., Knoxville, TN, UNITED STATES
Shi, Hang, Knoxville, TN, UNITED STATES
Zemel, Paula C., Knoxville, TN, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002192264	A1	20021219	<--
APPLICATION INFO.:	US 2001-17568	A1	20011214	(10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-654357, filed on 1 Sep 2000, GRANTED, Pat. No. US 6384087			
	Continuation-in-part of Ser. No. WO 2001-US27432, filed on 4 Sep 2001, UNKNOWN			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	SALIWANCHIK LLOYD & SALIWANCHIK, A PROFESSIONAL ASSOCIATION, 2421 N.W. 41ST STREET, SUITE A-1, GAINESVILLE, FL, 326066669			
NUMBER OF CLAIMS:	34			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	12 Drawing Page(s)			
LINE COUNT:	1438			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The subject invention provides **methods** of inducing the loss of adipose tissue by providing a diet high in calcium. In one aspect of the invention, the calcium is provided in the form of dairy **products**. In yet another aspect of the invention, calcium is provided in the form of a dietary supplement, such as calcium carbonate, of vitamin supplements. **Methods** of suppressing [Ca.sup.2+].sub.i levels in individuals are also provided. The subject invention also provides **methods** of stimulating lipolysis, inhibiting lipogenesis, and increasing the expression of white adipose tissue uncoupling protein 2 (UPC2). The subject invention also provides **methods** of increasing the core temperature of an individual.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 16 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2002:330260 USPATFULL

TITLE: Bioavailable composition of natural and

INVENTOR(S): **synthetic hca**
Majeed, Muhammed, Piscataway, NJ, UNITED STATES
Hadmaev, Vladimir, Piscataway, NJ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002187943	A1	20021212	<--
APPLICATION INFO.:	US 2002-926746	A1	20020606	(9)
	WO 2001-US41748		20010817	
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	ARENT FOX KINTNER PLOTKIN & KAHN, 1050 CONNECTICUT AVENUE, N.W., SUITE 400, WASHINGTON, DC, 20036			
NUMBER OF CLAIMS:	28			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	19 Drawing Page(s)			
LINE COUNT:	670			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to a composition comprising hydroxycitric acid (HCA) in combination with either one or both of garcinol and anthocyanin, and its use as a **weight-loss** therapy in animal subjects, preferably humans. The therapeutic effects for the composition observed in murine and human studies include a reduction in total body weight and body mass index, a reduction in body fat, an increase in **lean body mass** and content of body water, and a reduction in perceived appetite level. Another composition for use in **weight-loss** therapy is also described relating to **forskolin** in combination with either one or both of garcinol and anthocyanin. The anti-oxidant properties of garcinol are described as being enhanced in the presence of HCA and anthocyanin, and the combination of HCA, garcinol and anthocyanin is also shown to exert greater citrate lyase inhibiting properties than either compound alone. **Methods** of obtaining HCA, garcinol or anthocyanin, or a composition containing all three compounds, are described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 17 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2002:242833 USPATFULL

TITLE: Materials and **methods** for the treatment or prevention of **obesity**

INVENTOR(S): Zemel, Michael B., Knoxville, TN, UNITED STATES
Shi, Hang, Knoxville, TN, UNITED STATES
Zemel, Paula C., Knoxville, TN, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002132014	A1	20020919	<--
APPLICATION INFO.:	US 2002-66057	A1	20020131	(10)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 2000-654357, filed on 1 Sep 2000, GRANTED, Pat. No. US 6384087			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	SALIWANCHIK LLOYD & SALIWANCHIK, A PROFESSIONAL ASSOCIATION, 2421 N.W. 41ST STREET, SUITE A-1, GAINESVILLE, FL, 326066669			
NUMBER OF CLAIMS:	25			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	13 Drawing Page(s)			

LINE COUNT: 1064

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The subject invention provides **methods** of inducing the loss of adipose tissue by providing a diet high in calcium. In one aspect of the invention, the calcium is provided in the form of dairy **products**. In yet another aspect of the invention, calcium is provided in the form of a dietary supplement, such as calcium carbonate, of vitamin supplements. **Methods** of suppressing [Ca.sup.2+].sub.i levels in individuals are also provided. The subject invention also provides **methods** of stimulating lipolysis, inhibiting lipogenesis, and increasing the expression of white adipose tissue uncoupling protein 2 (UCP2). The subject invention also provides **methods** of increasing the core temperature of an individual.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 18 OF 19 USPATFULL on STN

ACCESSION NUMBER: 2002:102534 USPATFULL

TITLE: Materials and **methods** for the treatment or prevention of **obesity**

INVENTOR(S): Zemel, Michael B., Knoxville, TN, United States

Shi, Hang, Knoxville, TN, United States

Zemel, Paula C., Knoxville, TN, United States

PATENT ASSIGNEE(S): University of Tennessee Research Corporation, Inc.,
Knoxville, TN, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6384087	B1	20020507	<--
APPLICATION INFO.:	US 2000-654357		20000901	(9)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	GRANTED			
PRIMARY EXAMINER:	Webman, Edward J.			
LEGAL REPRESENTATIVE:	Saliwanchik, Lloyd & Saliwanchik			
NUMBER OF CLAIMS:	9			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	22 Drawing Figure(s); 12 Drawing Page(s)			
LINE COUNT:	1047			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The subject invention provides **methods** of inducing the loss of adipose tissue by providing a diet high in calcium. In one aspect of the invention, the calcium is provided in the form of dairy **products**. In yet another aspect of the invention, calcium is provided in the form of a dietary supplement, such as calcium carbonate, of vitamin supplements. **Methods** of suppressing [Ca.sup.2+].sub.i levels in individuals are also provided. The subject invention also provides **methods** of stimulating lipolysis, inhibiting lipogenesis, and increasing the expression of white adipose tissue uncoupling protein 2 (UCP2). The subject invention also provides **methods** of increasing the core temperature of an individual.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L26 ANSWER 19 OF 19 USPATFULL on STN

ACCESSION NUMBER: 1998:108427 USPATFULL

TITLE: **Method of preparing** a forskohlin
composition from forskohlin extract and use of
forskohlin for promoting **lean body**
mass and treating mood disorders

INVENTOR(S): Majeed, Muhammed, Piscataway, NJ, United States

PATENT ASSIGNEE(S): Badmaey, Viadimir, Piscataway, NJ, United States
Rajendran, R., Bangalora, India
Sabinsa Corporation, Piscataway, NJ, United States
(U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5804596		19980908	<--
APPLICATION INFO.:	US 1997-807652		19970227	(8)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Goldberg, Jerome D.			
LEGAL REPRESENTATIVE:	Nikaido Marmelstein Murray & Oram LLP			
NUMBER OF CLAIMS:	8			
EXEMPLARY CLAIM:	1			
LINE COUNT:	405			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A **method** of promoting **lean body mass** in an individual is disclosed, comprising administering to the individual a **lean body mass** promoting effective amount of forskohlin. A **method** of treating a mood disorder is also disclosed, comprising administering to a patient in need thereof a mood disorder treating effective amount of forskohlin. Compositions suitable for promoting **lean body mass** and/or treating a mood disorder are also disclosed, the composition comprising about 1 to about 40% forskohlin in combination with at least one physiologically acceptable carrier or excipient. A **method** of **preparing** a forskohlin composition from a forskohlin extract of Coleus Forskoli plant is further disclosed, as well as a forskohlin composition **prepared** by the **method**.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d ibib abs ind l12 1-4

L12 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:219534 HCAPLUS
DOCUMENT NUMBER: 142:285215
TITLE: Process for preparing water soluble **diterpenes**
and their applications
INVENTOR(S): **Majeed, Muhammed**; Kumar, Arvind;
Nagabhushanam, Kalyanam; **Prakash, Subbalakshmi**
PATENT ASSIGNEE(S): Sami Labs Limited, India
SOURCE: U.S. Pat. Appl. Publ., 7 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005051483	A1	20050310	US 2003-605086	20030908
US 6960300	B2	20051101		
WO 2005025500	A2	20050324	WO 2004-US28644	20040902
WO 2005025500	A3	20050609		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

US 2005284812 A1 20051229 US 2005-215040 20050831

PRIORITY APPLN. INFO.: US 2003-605086 A 20030908

AB Aqueous solns. of **diterpenes** such as Forskolin, its congeners,
analogs and derivs., up to approx. 6% concentration, are prepared using
suitably

substituted cyclodextrin as a solubilizing agents. In the absence of
cyclodextrin, some **diterpenes** such as Forskolin are soluble in
water only to concns. of about 0.001%. Such aqueous solns. find applications
in topical and systemic use, as pharmaceutical, cosmetic, nutritional
preps. containing **diterpenes** such as Forskolin and congeners. Thus
forskolin (98.5 % assay, 25 mg) was added to 1 mL water containing in the
dissolved state 500 mg hydroxypropyl β cyclodextrin, HPBCD, (50%); the
suspension was agitated at 75 RPM in an isothermal shaker for 60 h at
temperature 30°C. Resulting solution was filtered through 0.45 μ m nylon
filter and analyzed for the content of Forskolin by HPLC 1.33 mg/mL or
0.133 % w/v.

IC ICM B01D011-00

INCL 210634000; X42-447.5; X42-440.0; X21-080.6

CC 63-6 (Pharmaceuticals)

Section cross-reference(s): 17, 62

ST **diterpene** forskolin soly cyclodextran

IT Skin, disease

(aging; process for preparing water soluble **diterpenes** and their
applications)

IT Collagens, biological studies

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(booster; process for preparing water soluble **diterpenes** and their applications)

IT Skin
(cellulite, treatment; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(drops; process for preparing water soluble **diterpenes** and their applications)

IT Eye, disease
(dry eye syndrome; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(gels; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(injections, i.v.; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(injections, s.c.; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(lotions; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(ointments, creams; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(ophthalmic; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(oral; process for preparing water soluble **diterpenes** and their applications)

IT Allergy
Allergy inhibitors
Anti-inflammatory agents
Antihypertensives
Antiobesity agents
Antioxidants
Cosmetics
Dentifrices
Glaucoma (disease)
Human
Hypertension
Obesity
Plectranthus barbatus
Recrystallization
Solubility
Solubilizers
Solvents
(process for preparing water soluble **diterpenes** and their applications)

IT **Diterpenes**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(sprays; process for preparing water soluble **diterpenes** and their applications)

IT Drug delivery systems
(sublingual; process for preparing water soluble **diterpenes** and

- their applications)
- IT Diet
(supplements, beverages; process for preparing water soluble **diterpenes** and their applications)
- IT Drug delivery systems
(suspensions; process for preparing water soluble **diterpenes** and their applications)
- IT Drug delivery systems
(sustained-release; process for preparing water soluble **diterpenes** and their applications)
- IT Drug delivery systems
(topical; process for preparing water soluble **diterpenes** and their applications)
- IT Drugs
(veterinary; process for preparing water soluble **diterpenes** and their applications)
- IT 149064-55-1, Garcinol
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(combination with; process for preparing water soluble **diterpenes** and their applications)
- IT 6205-14-7, Hydroxycitric acid
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination with; process for preparing water soluble **diterpenes** and their applications)
- IT 9001-62-1, Lipase 9025-82-5, Phosphodiesterase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors, combination with; process for preparing water soluble **diterpenes** and their applications)
- IT 64-17-5, Ethanol, biological studies 67-64-1, Acetone, biological studies 75-09-2, Methylene chloride, biological studies 141-78-6, Ethyl acetate, biological studies 9003-39-8, PVP 9004-61-9, Hyaluronic acid
RL: NUU (Other use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(process for preparing water soluble **diterpenes** and their applications)
- IT 7585-39-9, β -Cyclodextrin 7585-39-9D, β -Cyclodextrin, randomly methylated, or 2-hydroxypropyl derivative 10016-20-3, α -Cyclodextrin 17465-86-0, γ -Cyclodextrin 17465-86-0D, γ -Cyclodextrin, 2-hydroxypropyl derivative 64657-20-1, 7-Deacetylforskolin 64657-21-2, Isoforskolin 66575-29-9, Forskolin
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(process for preparing water soluble **diterpenes** and their applications)

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:696074 HCAPLUS

DOCUMENT NUMBER: 137:222013

TITLE: Composition and methods containing an antimicrobial essential oil extracted *Coleus forskohlii*

INVENTOR(S): Majeed, Muhammed; Prakash, Subbalakshmi

PATENT ASSIGNEE(S): Sabinsa Corporation, USA

SOURCE: PCT Int. Appl., 24 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002070638	A2	20020912	WO 2002-US3391	20020220
WO 2002070638	A3	20031218		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2002160066	A1	20021031	US 2002-78262	20020220
US 6607712	B2	20030819		

PRIORITY APPLN. INFO.:

US 2001-269330P P 20010220

AB An essential oil composition from *C. forskohlii* is used in compns. and methods for the treatment of skin infections and in the prevention and treatment of dental caries. The essential oil is obtained by supercrit. extraction with carbon dioxide of *C. forskohlii* root material, purification of the essential oil by solvent extraction, removal of impurities by crystallization, and further

purification of the essential oil by distillation to obtain a purified composition

comprising at least 7.5% bornyl acetate, at least 3.5% 3-decanone, at least 3.75% of an azulene derivative, at least 1% α -pinene, and at least 0.75% β -pinene.

IC ICM C11B009-02

ICS A61K035-78

CC 63-4 (Pharmaceuticals)

Section cross-reference(s): 1, 62

ST essential oil *Coleus* antimicrobial oral topical skin infection

IT Infection

(cutaneous; preparation of antimicrobial essential oil composition extracted from

Coleus forskohlii)

IT Crystallization

(impurities removal by; preparation of antimicrobial essential oil composition

extracted from *Coleus forskohlii*)

IT Periodontium, disease

Skin, disease

(infection; preparation of antimicrobial essential oil composition extracted from

Coleus forskohlii)

IT *Candida albicans**Escherichia coli**Propionibacterium acnes**Staphylococcus aureus**Staphylococcus epidermidis**Streptococcus mutans*

(inhibition of; preparation of antimicrobial essential oil composition extracted from

Coleus forskohlii)

IT Drug delivery systems

(oral; preparation of antimicrobial essential oil composition extracted from *Coleus*

forskohlii)
IT Antibacterial agents
Antimicrobial agents
Fungicides
Plectranthus barbatus
Skin preparations (pharmaceutical)
(preparation of antimicrobial essential oil composition extracted from
Coleus forskohlii)
IT **Sesquiterpenes**
Terpenes, biological studies
RL: NPO (Natural product occurrence); BIOL (Biological study); OCCU
(Occurrence)
(preparation of antimicrobial essential oil composition extracted from
Coleus forskohlii)
IT Essential oils
RL: PUR (Purification or recovery); THU (Therapeutic use); BIOL
(Biological study); PREP (Preparation); USES (Uses)
(preparation of antimicrobial essential oil composition extracted from
Coleus forskohlii)
IT Extraction
(supercrit.; preparation of antimicrobial essential oil composition
extracted from
Coleus forskohlii)
IT Drug delivery systems
(topical; preparation of antimicrobial essential oil composition extracted
from
Coleus forskohlii)
IT 76-49-3, Bornyl acetate 80-56-8, α -Pinene 127-91-3,
 β -Pinene 275-51-4D, Azulene, derivs. 473-13-2, α -Selinene
489-40-7, α -Gurjunene 928-80-3, 3-Decanone 6753-98-6,
 α -Humulene
RL: NPO (Natural product occurrence); BIOL (Biological study); OCCU
(Occurrence)
(preparation of antimicrobial essential oil composition extracted from
Coleus forskohlii)
IT 124-38-9, Carbon dioxide, uses
RL: NUU (Other use, unclassified); USES (Uses)
(supercrit. extraction with; preparation of antimicrobial essential oil
composition
extracted from Coleus forskohlii)

L12 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:658137 HCAPLUS
DOCUMENT NUMBER: 137:190761
TITLE: Water soluble boswellic acids, their preparation and
use for treating inflammatory conditions
INVENTOR(S): **Majeed, Muhammed**; Badmaev, Vladimir
PATENT ASSIGNEE(S): Sabinsa Corporation, USA
SOURCE: PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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 WO 2002066491 A1 20020829 WO 2002-US3384 20020215
 W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
 CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
 GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
 LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
 PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
 UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
 CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
 BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 2001-268713P

P 20010215

AB A new composition, which can be formed through a method comprising: (a) dissolving mixts. of boswellic acids in a water and alc. solution to form a mixture; (b) adding one or more alkali salts to the mixture to form a salt solution; (c) filtering the solution to sep. un-reacted alkali salt from a filtrate; and (d) recovering the soluble boswellic acid mixture from the filtrate, is described. Addnl., the new composition can be formed by using super critical carbon dioxide. The new composition can be used to alleviate numerous inflammatory conditions, including, but not limited to, rheumatoid arthritis and osteoarthritis, colon cancer, prostate cancer and breast cancer, and a broad range of neurodegenerative conditions, such as Alzheimer's disease and Parkinson's disease. The composition can be administered parenterally, orally, or topically.

IC ICM C07J063-00

ICS A61K031-56; C07C051-41

CC 63-6 (Pharmaceuticals)

Section cross-reference(s): 1

ST boswellic acid prepn oral parenteral topical inflammation

IT **Triterpenes**

RL: PKT (Pharmacokinetics); PUR (Purification or recovery); THU
 (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES
 (Uses)

(carboxy; preparation of water-soluble boswellic acids for oral, parenteral,
 and topical administration for treatment of inflammation)

IT Intestine, neoplasm

(colon; preparation of water-soluble boswellic acids for oral, parenteral,

and

topical administration for treatment of inflammation)

IT Nervous system, disease

(degeneration; preparation of water-soluble boswellic acids for oral,
 parenteral, and topical administration for treatment of inflammation)

IT Filters

(nutsche; preparation of water-soluble boswellic acids for oral, parenteral,
 and topical administration for treatment of inflammation)

IT Resins

RL: CPS (Chemical process); PEP (Physical, engineering or chemical
 process); PYP (Physical process); PROC (Process)

(oleoresins; preparation of water-soluble boswellic acids for oral,

parenteral,

and topical administration for treatment of inflammation)

IT Drug delivery systems

(oral; preparation of water-soluble boswellic acids for oral, parenteral,

and

topical administration for treatment of inflammation)

IT Drug delivery systems

(parenterals; preparation of water-soluble boswellic acids for oral,
 parenteral, and topical administration for treatment of inflammation)

IT Alzheimer's disease

Anion exchangers

Anti-inflammatory agents
Filtration
Freeze drying
Inflammation
Mammary gland, neoplasm
Osteoarthritis
Parkinson's disease
Prostate gland, neoplasm
Rheumatoid arthritis

(preparation of water-soluble boswellic acids for oral, parenteral, and
topical
administration for treatment of inflammation)

IT Alkali metal salts

Charcoal

RL: NUU (Other use, unclassified); USES (Uses)

(preparation of water-soluble boswellic acids for oral, parenteral, and
topical
administration for treatment of inflammation)

IT Drug delivery systems

(topical; preparation of water-soluble boswellic acids for oral, parenteral,
and topical administration for treatment of inflammation)

IT Carboxylic acids, biological studies

RL: PKT (Pharmacokinetics); PUR (Purification or recovery); THU
(Therapeutic use); BIOL (Biological study); PREP (Preparation); USES
(Uses)

(**triterpene**; preparation of water-soluble boswellic acids for oral,
parenteral, and topical administration for treatment of inflammation)

IT Drying

(vacuum; preparation of water-soluble boswellic acids for oral, parenteral,
and
topical administration for treatment of inflammation)

IT 67-56-1, Methanol, uses 7440-09-7D, Potassium, salts

RL: NUU (Other use, unclassified); USES (Uses)

(preparation of water-soluble boswellic acids for oral, parenteral, and
topical
administration for treatment of inflammation)

IT 631-69-6P, β -Boswellic acid 5968-70-7P, Acetyl- β -boswellic
acid 17019-92-0P, 11-keto- β -Boswellic acid 67416-61-9P,
Acetyl-11-keto- β -boswellic acid

RL: PKT (Pharmacokinetics); PUR (Purification or recovery); THU
(Therapeutic use); BIOL (Biological study); PREP (Preparation); USES
(Uses)

(preparation of water-soluble boswellic acids for oral, parenteral, and
topical
administration for treatment of inflammation)

IT 124-38-9, Carbon dioxide, uses

RL: NUU (Other use, unclassified); USES (Uses)

(supercrit.; preparation of water-soluble boswellic acids for oral,
parenteral,
and topical administration for treatment of inflammation)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2002:435649 HCAPLUS

DOCUMENT NUMBER: 138:66544

TITLE: **Diterpene** forskolin (Coleus forskohlii,
Benth.): a possible new compound for reduction of body
weight by increasing lean body mass

AUTHOR(S): Badmaev, Vladimir; **Majeed, Muhammed**; Conte,

CORPORATE SOURCE: Anthony A.; Parker, John E.
SOURCE: Sabinsa Corporation, Piscataway, NJ, 08854, USA
NutraCos (2002), 1(2), 6-7
CODEN: NUTRCP
PUBLISHER: B5 srl
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An extract of Coleus forskohlii root, Benth. (Fam. Labiatae) standardized for diterpene forskolin was tested in an open-field study of six overweight but healthy women. During the 8-wk trial with 250 mg of the extract standardized for 10% forskolin, the mean values for body weight and fat content were significantly decreased, whereas lean body mass was considerably increased as compared to the baseline.
CC 1-11 (Pharmacology)
ST diterpene forskolin Coleus antiobesity
IT Antiobesity agents
Human
Plectranthus barbatus
(diterpene forskolin (Coleus forskohlii, Benth.): a possible new compound for reduction of body weight by increasing lean body mass)
IT 66575-29-9, Forskolin
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(diterpene forskolin (Coleus forskohlii, Benth.): a possible new compound for reduction of body weight by increasing lean body mass)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT